In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 18-2V
Filed: May 9, 2025

MARILYN DATTE,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Robert Krakow, Law Office of Robert J. Krakow, P.C., New York, New York, for petitioner.
Jennifer Leigh Reynaud, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

On January 2, 2018, petitioner, Marilyn Datte, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that she suffered Guillain-Barré Syndrome ("GBS") caused-in-fact by a pneumococcal conjugate (Prevnar 13) vaccination she received on January 15, 2015. (ECF No. 1.) For the reasons set forth below, I conclude that petitioner is entitled to an award of compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations,

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] Within this ruling, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury.

In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B). In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In that context, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

GBS is a Table injury if onset occurs 3-42 days following receipt of a flu vaccine. 42 C.F.R. § 100.3(a)(XIV)(D). However, GBS is not a Table Injury relative to the Prevnar 13 vaccine at issue in this case. 42 C.F.R. § 100.3(a)(XII). To succeed on a claim that petitioner's Prevnar 13 vaccine caused GBS, petitioner must satisfy the burden of proof for "causation-in-fact."

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" with the logical sequence being supported by "reputable medical or scientific explanation." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for

the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. *Id*.

A petitioner may not receive a Vaccine Program award based solely on his or her assertions, but may support the petition with either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa-13(b)(1). A petitioner may rely upon circumstantial evidence. *Althen*, 418 F.3d at 1280. In that regard, the *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3. Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 300aa-13(b)(1)(A). The special master is required to consider the entirety of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

This case was originally assigned to another special master. (ECF No. 4.) Petitioner filed medical records, medical literature, a declaration, and an affidavit regarding damages (ECF Nos. 5-10, 15, 18; Exs. 1-20) and then filed her statement of completion in February of 2018. (ECF No. 19.) In November of 2018, respondent filed his Rule 4(c) Report, recommending against compensation. (ECF No. 30.) Petitioner

filed an expert report by neuroimmunologist, Lawrence Steinman, M.D., the following April. (ECF Nos. 36-38; Exs. 22-47.) This case was subsequently reassigned to undersigned in August of 2019. (ECF Nos. 42-43.)

In October of 2019, respondent filed expert reports from neurologist, Vinay Chaudhry, M.D., and immunologist, Robert Fujinami, Ph.D. (ECF Nos. 45-46; Ex. A-D.) Petitioner filed additional medical records in January and March of 2020. (ECF Nos. 49, 53; Ex. 48-49.) From September of 2020 to April of 2021, the parties engaged in two additional rounds of expert reports. (ECF Nos. 63-65, 68-69, 72, 78; Exs. 50-77, E-G.) Thereafter, a two-day entitlement hearing was scheduled to commence on February 7, 2023. (ECF No. 83.) Petitioner subsequently filed updated medical records. (ECF Nos. 86, 89, 91, 94, 96; Exs. 78-95.)

In September of 2022, respondent filed a joint status report on behalf of the parties, agreeing that this case was ripe for the previously scheduled entitlement hearing. (ECF No. 100.) However, two months later, petitioner filed a motion for leave to file additional exhibits. (ECF No. 102; Exs. 96-104.) Petitioner was permitted to file the exhibits (ECF No. 103), but the entitlement hearing was rescheduled to commence on April 4, 2023. (ECF No. 106.) Respondent then filed a supplemental report from Dr. Fujinami on March 1, 2023. (ECF No. 113; Ex. H.) Following the prehearing status conference on March 29, 2023, the parties each filed further additional medical literature. (ECF Nos. 125, 127; Exs. 108; I.)

A two-day entitlement hearing was held remotely via Zoom on April 4th and 5th, 2023. (*See* Transcript of Proceedings ("Tr"), at ECF Nos. 129-30.) During the hearing, petitioner presented testimony from Dr. Steinman and respondent presented testimony from Dr. Fujinami. (*See id.*) Following the hearing, I ordered the parties to file post-hearing briefs, and the parties completed their filings on December 7, 2023. (ECF Nos. 132, 135, 138.) This case is now ripe for a decision on entitlement.

III. Factual History

a. As reflected in the medical records

On January 15, 2015, petitioner received the subject Prevnar 13 vaccine. (Ex. 16.) She was 65 years old at the time of vaccination, and her past medical history was significant for type II diabetes, fatty liver disease, hypertension, glaucoma, hypothyroidism, anxiety, and obstructive sleep apnea. (Ex. 10, pp. 25-27, 31-33, 37-44, 47-52.) No new significant health issues were noted during the January 15, 2015 encounter. (*Id.* at 18-22.)

Ten days later, on January 25, 2015, petitioner presented to the emergency department with complaints of tingling in both her hands and feet. (Ex. 9, p. 181.) She was diagnosed with "new onset peripheral neuropathy/diabetes" and discharged with instructions to follow up with her primary care physician. (*Id.* at 173-74, 181.) The next day, petitioner returned to the emergency department with the same complaints of

tingling in her hands and feet. (*Id.* at 213.) She further complained of progressive numbness in her hands and feet and weakness, reporting that she was experiencing difficulties raising either arm in the air. (*Id.* at 213, 227.) Additionally, she presented with a "[p]etechiae like rash" on her face and redness in both sclera. (*Id.* at 227.) A CT scan of her brain and MRI of the brain and cervical spine were unrevealing. (*Id.* at 255, 264-66.) Petitioner was again discharged, but this time with a neurology referral. (*Id.* at 218-21.)

However, petitioner's condition continued to progress. After returning home following her discharge from the emergency room, petitioner reported experiencing pain in her neck, head, and sinuses as well as an unsteady gait. (Ex. 10, p. 88.) As the day progressed, she lost her sense of taste, started to experience tingling in her tongue, and began to have no strength in her arms and legs. (*Id.*) The following morning, on January 27, 2015, petitioner reported waking up around 5 a.m. with a headache, vomiting, and difficulty swallowing. (*Id.* at 89.) She was so weak that she could not stand or move her arms or legs. (*Id.*) At this point, petitioner decided she needed to return to the emergency room. (*Id.*) However, due to her extreme weakness, petitioner called EMS and was transported by ambulance to MidMichigan Medical Center - Midland. (*Id.*; Ex. 9, p. 407, 411.) Upon arrival, petitioner was extremely weak in her upper and lower limbs and had dysphagia, shortness of breath, and accelerated hypertension. (Ex. 9, p. 407, 411; Ex. 10, p. 89; Ex. 12, p. 45.) She was immediately transferred to the intensive care unit for further treatment. (Ex. 10, p. 89.) Her treating providers noted a high clinical suspicion for GBS. (*Id.* at 80, 90; Ex. 12, p. 47.) Petitioner's treating neurologist, Gregory Dardas, M.D., indicated that she "recently had a pneumonia vaccine but otherwise has been in her usual state of health." (Ex. 10, pp. 79-80.) By January 28, 2015, petitioner's working diagnosis was acute inflammatory demyelinating polyradicular neuropathy. (Ex. 9, p. 352; Ex. 12, p. 43.) Dr. Dardas noted that petitioner was "quadriparetic but not quadriplegic." (Ex. 12, pp. 43-44.) A nerve conduction study performed on January 29, 2015, confirmed diffuse axonal and demyelinating polyneuropathy consistent with GBS. (Ex. 9, p. 937.) Petitioner's treating pulmonologist concurred in diagnosis, assessing petitioner with acute respiratory failure due to ascending paralysis consistent with the acute inflammatory demyelinating polyneuropathy ("AIDP") form of GBS. (Ex. 12, pp. 21-22.) The pulmonologist also noted that petitioner's presentation of hypertension with "wide fluctuations" was consistent with GBS induced autonomic dysfunction. (*Id.* at 22.)

During her hospitalization, she underwent a lumbar puncture, which was negative, and a five-day course of IVIG treatment. (Ex., 10, pp. 79-80, 90; Ex. 9, pp. 335, 348; Ex. 12, pp. 33-34, 47.) She also completed various therapies to regain functionality. (Ex. 9, pp. 1044-52.) Despite these treatments, petitioner's condition continued to deteriorate. (Ex. 12, p. 34.) Petitioner experienced acute respiratory failure that was attributed to "acute inflammatory demyelinating polyneuropathy status post IVIG." (Ex. 9, p. 340.) As a result, she was intubated and placed on a mechanical ventilator. (*Id.* at 348; Ex. 12, pp. 36-38.) Due to an inability to wean off the ventilator, petitioner required a tracheostomy and a percutaneous endoscopic gastrostomy, as well as a bronchoscopy for mucus plugging and atelectasis of the left lower lung. (Ex. 9, pp.

327-29, 337-38, 1034; Ex. 12, pp. 34, 37.) On February 8, 2015, physicians observed that petitioner could shrug her shoulders and noted a "flicker of movement" in both hands and feet. (Ex. 9, p. 338.) The next day, on February 9, 2015, her neurologist documented that while petitioner had shown some signs of modest improvement, "she certainly has quite a ways to go." (Ex. 12, pp. 39-40.) He noted that she was able to move her hands and feet on command and could "move with gravity," but she could not lift her head or limbs off the bed. (*Id.* at 39; Ex. 9, p. 336.) Facial weakness and lack of sensation to pinprick were also noted. (Ex. 12, p. 39.)

On February 10, 2015, petitioner was discharged from MidMichigan Medical Center and transported to Select Specialty Hospital for long-term acute care. (Ex. 2, pp. 39-40; Ex. 12, pp. 33-38.) On discharge, petitioner was still bedridden, struggling with weakness, and required ventilation; however, she was noted to be making some progress and able to move all extremities. (Ex. 12, pp. 34, 37.) Subsequent chest x-rays showed additional density and pulmonary congestion in her right lung, which was attributed to pneumonia. (Ex. 2, pp. 997, 1000; Ex. 4, pp. 80, 82, 84.) Then, on February 13, 2015, petitioner was admitted to the intensive care unit at Covenant Hospital after she experienced severe respiratory distress and became unresponsive due to hypotension. (Ex. 2, pp. 30, 32; Ex. 18, pp. 10, 16, 64-67.) She was treated for septic shock, SIRS, pneumonia, urinary tract infection ("UTI") with methicillin-resistant *Staphylococcus epidermidis* ("MRSE"), an infection at her tracheostomy site, as well as acute pancreatitis. (Ex. 18, pp.16-17.) On February 19, 2015, petitioner returned to Select Specialty Hospital. (*Id.* at 16-18; Ex. 2, pp. 30, 32.) During her stay, she saw speech-language pathologists to work on swallowing and tongue strength. (Ex. 2, pp. 793-96.) She also had her tracheostomy removed. (Ex. 20, p. 406.) Physician notes indicate that her extremity weakness was improving, although she continued to struggle with blood pressure fluctuation, pneumonia, bowel issues, and UTI. (Ex. 4, pp. 4-52.)

By March 17, 2015, petitioner was cleared for discharge and transferred to Huron Woods Nursing Center for occupational and physical therapy. (Ex. 2, pp. 27-28; Ex. 4, pp. 3-5; Ex. 20, pp. 406-08.) A physical examination showed that petitioner was unable to move her lower extremities or to turnover/sit up on her own. (Ex. 20, p. 408.) She also complained of muscle pain and weakness. (*Id.* at 407-08.) She worked on functional activities, such as hygiene and toileting, mobility, balancing, and strengthening. (*Id.* at 185.) Petitioner made substantial progress in regaining strength and functional activities during rehabilitation. (*Id.* at 129.) During this rehabilitation, on April 2, 2015, petitioner was diagnosed with deep vein thrombosis in her right leg and was transferred to Covenant Hospital again for treatment for two days. (Ex. 18, pp. 543-45, 552.) Then, on April 19, 2015, petitioner was sent to McLaren Medical Center due to a repeat episode of supraventricular tachycardia. (Ex. 20, p. 79.) She was discharged the same day and returned to Huron Woods. (*Id.*)

Petitioner was discharged home from Huron Woods on May 29, 2015. (Ex. 20, pp. 358, 367.) She was discharged home with a walker and wheelchair. (*Id.* at 367.) Petitioner's discharge summary noted that she was ambulatory with a walker and minimal to moderate assistance. (*Id.* at 368.) Additionally, the discharge summary

indicated that petitioner could complete most of her activities of daily living with minimal to moderate assistance. (*Id.*) Petitioner continued with in-home physical and occupational therapy. (Ex. 10, pp. 13-14; Ex. 5, p. 46; Ex. 13, p. 52.)

On June 1, 2015, petitioner followed up with her primary care provider. (Ex. 10, p. 13.) At this visit, her primary care provider reported that petitioner was navigating short distances with a walker. (*Id.*) However, her primary care provider documented that petitioner was unable to walk with her walker independently, noting that she required a one person assist. (*Id.*) She had ceased taking her Neurontin medication for paresthesia due to nausea and diarrhea. (*Id.*) While she was still experiencing weakness, most of her weakness was now in her feet. (*Id.* at 13, 15.) Notably, the history of present illness indicates that petitioner's GBS "started 10 days after getting the prevnar 13" vaccine. (*Id.* at 13.) Petitioner's physician assessed her with "Guillain-Barre syndrome following vaccination." (*Id.* at 15.)

From July 14 through August 25, 2015, petitioner participated in physical therapy at Auburn Physical Therapy. (Ex. 5, p. 2.) She worked on strength, endurance, stability, transfers, bed mobility, gait training, balance, and postural training. (*Id.* at 7.) Upon discharge, petitioner reported "85-90% improvement." (*Id.* at 2.) She reported being able to ambulate independently at home and walk for about one hour with a cane, although she was limited in her ability to ambulate on uneven surfaces or play with her grandchildren. (*Id.* at 2.)

At a follow up exam on October 15, 2015, petitioner indicated that she was doing well and ambulating with a cane. (Ex. 10, p. 1.) Petitioner's primary care provider indicated that she continued to show slow improvement, although her balance was a little off and she still had "prickling in hands and feet." (*Id.*) On March 8, 2016, petitioner's neurologist indicated that her GBS was "more or less completely resolved." (Ex. 12, pp. 12-15.)

As of March 16, 2017, "Guillain-Barre syndrome following vaccination was still listed as one petitioner's active problems. (Ex. 13, p. 28.) Petitioner was noted to be experiencing continued numbness in both feet at this time; however, it was unclear whether this symptom was due to GBS or petitioner's preexisting diabetes myelitis. (*Id.*) On September 26, 2017, petitioner presented to her neurologist, reporting issues with her feet, including plantar fascia pain, that worsened when she was standing or walking. (Ex. 12, pp. 6-7.) Petitioner's presentation suggested that her symptoms were the result of "something other than diabetic neuropathic pain." (*Id.* at 6.) The EMG and nerve conduction study were "notable for diffuse axonal and to a lesser extent demyelinating polyneuropathy." (*Id.* at 7.) Petitioner's neurologist noted the difficulty in ascertaining the chronicity of her presentation and opined that the clinical findings were likely related to her history of GBS and diabetes. (*Id.*)

Petitioner's primary care provider continued to document her GBS as an active problem in 2020 and 2021. (Ex. 78, pp. 8, 14, 19, 23, 28; Ex. 48, p. 1.) At an encounter on March 31, 2021, her primary care provider noted that petitioner still experiences neuropathy in both feet from her GBS. (Ex. 78, p. 27.)

b. Petitioner's declaration

Petitioner declares that, prior to the subject vaccination, she was generally healthy, her diabetes was under control, though she was monitored for hypertension and several other conditions. (Ex. 17, ¶ 5.) Petitioner was administered a Prevnar 13 vaccine on January 15, 2015, during an appointment for her annual physical. (*Id.* ¶¶ 4, 6-8.)

According to petitioner, she noticed tingling in her fingers and toes while walking on the treadmill in the late afternoon of January 25, 2015. (Ex. 17, ¶ 9.) Petitioner's husband transported her to the emergency department at Mid-Michigan Medical Center in Midland. (*Id.* ¶ 10.) She was diagnosed with diabetic neuropathy and sent home with a prescription. (*Id.* ¶ 11.) Petitioner declares the next day, January 26, 2015, she awoke with tingling extending to her feet and hands. (*Id.* ¶ 12.) She also describes difficulty walking, prompting her husband to take her back to the emergency department a second time. (*Id.*) Petitioner declares she was sent home with no prescribed medications. (*Id.* ¶ 14.) An appointment was scheduled with neurologist Dr. Gregory Dardas. (*Id.* ¶ 13.)

On January 27, 2015, petitioner describes difficulty standing and walking, requiring assistance to bathroom. (Ex. 17, ¶ 15.) Petitioner declares her husband called an ambulance and she was transported to the Mid-Michigan emergency department for the third time where she was subsequently admitted to the ICU. (*Id.* ¶¶ 15-16.) Petitioner recalls "I was unable to move most of my body. I was unable to walk or stand. I felt numb all over and had trouble breathing and swallowing." (*Id.* ¶ 16.) Petitioner was started on IVIG treatment for GBS. (*Id.* ¶ 17.) On January 29, 2015, petitioner was put on a ventilator. (*Id.* ¶ 18.) On January 31, 2015, petitioner underwent her last IVIG treatment. (*Id.* ¶ 19.) She recalls, "I was semi-conscious at times." (*Id.*) On February 5, 2015, petitioner underwent surgery to place a feeding tube and tracheostomy. (*Id.* ¶ 20.)

Petitioner recalls that she started to experience signs of slow improvement while at Select Specialty Hospital. (Ex. 17, ¶ 25.) Once petitioner was discharged to Huron Woods, she started physical and occupational therapy. (*Id.* ¶ 26.) She remembers that she was first able to attempt to stand in April of 2015. (*Id.*) By the end of May of 2015, petitioner recalls that she experienced progress with walking. (*Id.*) After being discharged from Huron Woods, petitioner continued her rehabilitation with in-home physical and occupational therapy. (*Id.* ¶ 27.) By the end of June of 2015, petitioner avers being able to walk short distances with the assistance of a cane. (*Id.*) In July of 2015, petitioner's in-home physical and occupational therapy concluded, and she started to work with outpatient physical therapy through the end of August of 2015. (*Id.* ¶¶ 27-28.) At the time of filing her declaration in January of 2018, petitioner continued to experience pain and discomfort in her feet and fingertips. (*Id.* ¶ 29.) She avers that her continuing symptoms worsen when she is on her feet but that the pain and discomfort subside when she is sitting or lying down. (*Id.*)

IV. Summary of Expert Opinions

a. Petitioner's Expert, Lawrence Steinman, M.D.[3]

Petitioner filed three expert reports and a letter from Dr. Steinman. (Exs. 22, 50, 75, 96.) Dr. Steinman also testified at the hearing. (Tr. 5.) He opined that petitioner's GBS was caused by her January 15, 2015, Prevnar 13 vaccination. (*Id.* at 36-37; Ex. 22, pp. 1, 22.) Dr. Steinman identified two molecular mimics relevant to GBS in the composition of the Prevnar 13 vaccine, one relating to phosphoglycerol within the pneumococcal polysaccharide antigen, and a second from the CRM protein conjugate.[4] (Ex. 22, p. 12; Tr. 43.)

Initially, Dr. Steinman sought to determine if the Prevnar 13 vaccine contained phospholipids, which he could not confirm. (Ex. 22, p. 14; Tr. 63-64 (citing Prescribing Information, Prevnar 13 (Pneumococcal 13-Valent Conjugate Vaccine [Diphtheria CRM_{197} Protein]): Suspension for Intramuscular Injection [hereinafter Prevnar 13 Package Insert] (Ex. 15; *see also* Ex. 31)); Ex. 75, p. 5.) However, he explained that the patent for the vaccine confirms that the Prevnar 13 vaccine contains phosphoglycerol linkages, which he opined are supportive of his causal theory. (Ex. 75, pp. 5-8 (citing U.S. Patent No. 9,492,559 B2 (issued Nov. 15, 2016) [hereinafter Prevnar 13 Patent] (Ex. 51)); Tr. 65-67.) That is, Dr. Steinman clarified during the hearing that the vaccine contains phosphoglycerol, which he indicated is a component of phospholipids, not phospholipids.[5] (Tr. 43, 90, 161.) Upon his review of Chuang et al., Dr. Steinman determined that the 19A strain of *S. pneumoniae* contains an expression of phosphorylcholine, which plays a key role in the pathophysiology of pneumonia infection. (Ex. 22, p. 14 (citing Yi-Ping Chuang et al., *Impact of the* glpQ2 *Gene of Virulence in a* Streptococcus Pneumoniae *Serotype 19A Sequence Type 320 Strain*, 83 INFECTION & IMMUNITY 682 (2015) (Ex. 36)).) The authors explain that surface phosphorylcholine expression in *S. pneumoniae* 19AST320 during the exponential phase "contributes to the severity of pneumonia by promoting adherence and host cell cytotoxicity." (*Id.* (quoting Chuang et al., *supra*, at Ex. 36, p. 1).) Dr. Steinman thus

[3] Dr. Steinman received his medical degree from Harvard in 1973. (Ex. 23.) He completed an internship and residency at Stanford University Hospital in 1973 and 1947, respectively, before going on to complete a neuroimmunology fellowship at the Weizmann Institute of Science in 1977. (*Id.* at 1; Tr. 10.) He returned to Stanford university to complete an additional neurology residency in 1980. (Ex. 23, p. 1; Tr. 10.) Since then, Dr. Steinman has worked as a professor in the neurology department at Stanford University. (Ex. 22, p. 1; Tr. 5, 10.) In his clinical capacity, Dr. Steinman has treated both adult and pediatric patients who suffered from various forms of autoimmune disease of the nervous system, and he has diagnosed hundreds of GBS patients. (Ex. 22, p. 1; Tr. 6-9.) In his research capacity, Dr. Steinman's has focused on how the immune system attacks the nervous system, and he has published on the subject of molecular mimicry. (Ex. 22, pp. 1-4; Ex. 23, pp. 5-45; Tr. 11-12, 14-15.) Dr. Steinman is board certified in neurology. (Ex. 23, p. 2; Tr. 17.) At the hearing, Dr. Steinman was proffered as an expert in neurology and neuroimmunology without objection. (Tr. 26, 30-31.)

[4] Because this ruling resolves entitlement in petitioner's favor based on the phosphoglycerol theory, the CRM protein theory is not discussed as it is not necessary to reach that theory.

[5] Respondent asserts that this change in Dr. Steinman's approach should be viewed as discrediting. (ECF No. 135, pp. 23-25.)

concluded the Prevnar 13 vaccine could immunize against this component of pneumococcus. (*Id.* at 15 (citing Jennifer L. Kanter et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation*, 12 NATURE MED. 138 (2006) (Ex. 33); Peggy P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath that Resolve Neuroinflammation*, 4 SCI. TRANSLATIONAL MED. 1 (2012) (Ex. 34); Denong Wang et al., *Uncovering Cryptic Glycan Markers in Multiple Sclerosis (MS) and Experimental Autoimmune Encephalitis (EAE)*, 75 DRUG DEV. RSCH. 172 (2014) (Ex. 35).) He explained that the enzyme for metabolizing lipids and producing phosphorylcholine is present in strains 3, 6B, 19A and 19F. (*Id.* (citing Chuang et al., *supra*, at Ex. 36).) Moreover, strains 3, 19A, and 19F are present in Prevnar 13. (*Id.* (citing Prevnar 13 Package Insert, *supra*, at Ex. 15); Ex. 50, pp. 7, 18 (citing Prevnar 13 Patent, *supra*, at Ex. 51).) Dr. Steinman further noted that the vaccine contains 18C and 23F, both of which contain phosphoglycerol. (Ex. 50, pp. 7, 18; Tr. 71-72, 78, 311.)

Citing his own research, Dr. Steinman explained that phospholipids (and therefore phosphoglycerol) are components of myelin sheath, and they are targeted by antibodies in neuroinflammation. (Ex. 22, p. 13 (citing Kanter et al., *supra*, at Ex. 33; Ho et al., *supra*, at Ex. 34; Wang et al., *supra*, at Ex. 35); Tr. 43, 97, 298-99.) In Ho et al., Dr. Steinman and his colleagues demonstrated that "[l]ipids constitute 70% of the myelin sheath, and autoantibodies against lipids may contribute to the demyelination that characterizes multiple sclerosis." (Ex. 22, p. 13 (quoting Ho et al., *supra*, at Ex. 34).) The authors explained that they used lipid antigen microarrays and lipid mass spectrometry to identify lipid targets of the autoimmune response in an MS brain and in an animal model of MS. (*Id.*) Ho et al. found that autoantibodies in MS target a phosphate group in phosphatidylserine and oxidized phosphatidylcholine derivatives. (*Id.*; Tr. 97-98, 303-04.) The main target of the human immune response to the myelin lipids was the phosphoglycerol component – common to phosphatidyl-ethanolamine, phosphatidylcholine, phosphatidylserine. (Ex. 50, p. 4 (citing Ex. 22).)

Dr. Steinman also observed that antibodies to phospholipids are seen in GBS. (Ex. 22, p. 13 (citing B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with the Guillain-Barre Syndrome: Cross-Reactive or Pathogenic?*, 16 AUTOIMMUNITY 23 (1993) (Ex. 32)).) Prior to the Ho et al. study, Gilburd et al. published results demonstrating that the sera of 6 out of 16 GBS patients had autoantibodies to one or more of the antigens studied (phosphatidyl-ethanolamine, phosphatidylcholine, and phosphatidylserine). (*Id*. (citing Gilburd et al., *supra*, at Ex. 32).) Dr. Steinman stressed that two of the six patients tested had reactivity to phosphatidylserine or phosphatidylcholine. (*Id.*) Dr. Steinman explained that the structure of phosphatidylcholine and phosphatidylserine have in common a polar head group targeted by antibodies. (*Id.* at 14 (citing Ho et al., *supra*, at Ex. 34; Tr. 82-83).) He stressed that the phospholipids discussed in the Gilburd et al. paper all share a common phosphoglycerol group. (Ex. 50, pp. 4, 17.) This is the component of the phospholipids that is attacked by the immune system in MS – as shown in the later paper by Ho et al. (*Id.* at 4.) He acknowledged that petitioner's theory of causation relies on a scientific analogy to the documented MS immune response. (*Id.*) Dr. Steinman opined, however, that the findings in the Gilburd et al. paper are clarified by

application of the later findings by Ho et al., allowing a reinterpretation of the Gilburd et al. study's conclusion, which "can all be explained by the same immune response in each disease to a common structure." (*Id.*) In particular, in light of the Ho et al. paper, Dr. Steinman stressed the authors of the 1993 Gilburd et al. paper would have been "able to reinterpret and correct their assertion that these immune responses were the 'effect' and not the 'cause.'" (*Id.*)

Additionally, Dr. Steinman observed that in Nakos et al., phospholipid antibodies were found in patients with GBS, similar to the Gilburd et al. study's findings. (Ex. 50, pp. 11-12 (citing G. Nakos et al., *Anti-Phospholipid Antibodies in Serum from Patients with Guillain-Barré Syndrome*, 31 INTENSIVE CARE MED. 1401 (2005) (Ex. 55)).) In fact, four blood samples were obtained before and after treatment and tested for antibodies to phosphatidylcholine, phosphatidyl-inositol, cardiolipin, phosphatidic acid, phosphatidylserine, phosphatidylglycerol, phosphatidylethanolamine, sphingomyelin, and gangliosides. (*Id.* (citing Nakos et al., *supra*, at Ex. 55); Tr. 86-88, 315-20.) Antiphospholipid antibodies of the IgM, IgA, and IgG families were detected in all GBS patients but in none of the controls. (Ex. 50, p. 12 (citing Nakos et al., *supra*, at Ex. 55).) Phosphatidylinositol, cardiolipin, phosphatidylcholine, and phosphatidic acid were the main antigens, with cardiolipins presenting within one day of infusion. (*Id.*; Tr. 87-88, 316-18.) Accordingly, Dr. Steinman opined that Prevnar 13 could immunize against this phosphoglycerol component of pneumococcus common to the linkage for the polysaccharides in the vaccine. (Ex. 50, p. 12.)

Dr. Steinman opined that the common structure is phosphoglycerol. (Ex. 50, p. 4.) The Ho et al. paper indicates that the polar head group in phosphoglycerol and phosphocholine are the main targets of immune attack in MS patients. (*Id.* at 8 (citing Ho et al., *supra*, at Ex. 34).) Ho et al. observed that the saturated, nonpolar side chains mediate the protective effects of the therapeutic lipids; and the palmitic acid (representative of such fatty acid side chains present within the lipids) was able to reproduce the therapeutic effects of the lipids in vitro and in vivo. (*Id.* at 21 (quoting Ho et al., *supra*, at Ex. 34, p. 9); Tr. 84.) Yet, while the fatty acid side chains are the protective moiety, Dr. Steinman maintains that the phosphoglycerol is the target of the antibody. (Ex. 50, p. 21; Tr. 86 (discussing Nakos et al., *supra*, at Ex. 55), 90 (citing Ho et al., *supra*, at Ex. 34).) Dr. Steinman cited Barbar et al., which showed that "antibodies can have exquisite specificity for the polar head group, although they may bind with different affinities (strengths) depending on the bigger carrier molecule connected to the phosphate head group." (Ex. 96, p. 5 (citing Elisar Barbar et al., *Binding of Phenylphosphocholine–Carrier Conjugates to the Combining Site of Antibodies Maintains a Conformation of the Hapten*, 35 BIOCHEMISTRY 2958 (1996) (Ex. 102)).) He further cites Bryson et al., which found that immunization with a pneumococcal vaccine containing 23F makes an antibody that recognizes the polar head group on phosphoglycerol. (Tr. 96, 311 (citing Steve Bryson et al., *Structures of Preferred Human IgV Genes – Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation*, 196 J. IMMUNOLOGY 4723 (2016) (Ex. 103)).)

Dr. Steinman drew further support from the Chang et al. paper demonstrating that a phospholipid linkage is "quite necessary" for immunogenicity of the capsular polysaccharides. (Ex. 50, p. 10 (citing Janoi Chang et al., *Relevance of O-acetyl and Phosphoglycerol Groups for Antigenicity of* Streptococcus Pneumoniae *Serotype 18C Capsular Polysaccharide*, 30 VACCINE 7090 (2012) (Ex. 52)); Tr. 72-74, 76-77.) The authors explain that serotype 18C contains a repeating unit that has a complex structure – "a branched pentasaccharide with two apparently labile substituents: glycerol-phosphate and O-acetyl group." (Ex. 50, p. 10 (quoting Chang et al., *supra*, at Ex. 52); Tr. 72-73.) The "loss of these groups may potentially reduce the ability of the 18C polysaccharide to induce the desired immune response," thus the authors conclude the glycerol-phosphate "must be preserved for conserving adequate antigenicity of the 18C capsular polysaccharide." (Ex. 50, p. 10 (quoting Chang et al., *supra*, at Ex. 52); Tr. 72-74.) Lugowski et al. further suggests the Glycerol-1-P-OH is critical for the linkage to the 18C polysaccharide. (Ex. 50, pp. 10-11 (citing Czeslaw Lugowski & Harold Jennings, *Structural Determination of the Capsular Polysaccharide of* Streptococcus Pneumoniae *Type 18C (56)*, 131 CARBOHYDRATE RSCH. 119 (1984) (Ex. 53)).)

Dr. Steinman additionally proposed that alum adjuvant in the Prevnar 13 vaccine can lead to an increase in pro-inflammatory cytokines, such as IL-1 and IL-18, and contribute to the pathogenesis of GBS. (Ex. 50, pp. 23-25.) He maintains IL-1 and IL-18 are strongly upregulated during active GBS and its animal models but reduce as GBS resolves. (*Id.* at 23 (citing Rosetta Pedotti et al., *Severe Anaphylactic Reactions to Glutamic Acid Decarboxylase (GAD) Self Peptides in NOD Mice that Spontaneously Develop Autoimmune Type I Diabetes Mellitus*, 4 BMC IMMUNOLOGY 1 (2003) (Ex. 59); Stefanie Kuerten & Paul V. Lehmann, *The Immune Pathogenesis of Experimental Autoimmune Encephalomyelitis: Lessons Learned for Multiple Sclerosis*, 31 J. INTERFERON & CYTOKINE RSCH. 907 (2011) (Ex. 60); Ludwig Kappos et al., *Induction of a Non-Encephalitogenic Type 2 T Helper-Cell Autoimmune Response in Multiples Sclerosis After Administration of an Altered Peptide Ligand in a Placebo-Controlled, Randomized Phase II Trial*, 6 NATURE MED. 1176 (2000) (Ex. 61); Stephanie Eisenbarth et al., *Crucial Role for the Nalp3 Inflammasome in the Immunostimulatory Properties of Aluminum Adjuvants*, 453 NATURE 1122 (2008) (Ex. 62); Anna Sokolovska et al., *Activation of Dendritic Cells and Induction of CD4+ T Cell Differentiation by Aluminum-Containing Adjuvants*, 25 VACCINE 4575 (2007) (Ex. 63); J.W. Mannhalter et al., *Modulation of the Human Immune Response by the Non-Toxic and Non-Pyrogenic Adjuvant Aluminum Hydroxide: Effect on Antigen Uptake and Antigen Presentation*, 61 CLINICAL & EXPERIMENTAL IMMUNOLOGY 143 (1985) (Ex. 64)).) Jander and Guido reported that IL-18 serum levels were significantly higher in GBS patients than in noninflammatory neurologic disease controls – thus "implicating the Th1-inducing cytokine IL-18 in the pathogenesis of acute immune-mediated PNS demyelination." (*Id.* at 23-24 (quoting Sebastian Jander & Guido Stoll, *Interleukin-18 is Induced in Acute Inflammatory Demyelinating Polyneuropathy,* 114 J. NEUROIMMUNOLogy 253 (2001) (Ex. 67)).) On the whole, Dr. Steinman opined these papers constitute strong evidence for how the pneumococcal vaccine containing alum as an adjuvant would lead to inflammatory polyneuropathy. (*Id.* at 24.) However, Dr. Steinman stated during the

hearing that his opinion regarding any pro-inflammatory role played by the alum adjuvant is not an essential component of his causal theory. (Tr. 115-16, 118.)

Dr. Steinman cited two reports of GBS following pneumococcal vaccination. (Ex. 75, p. 10 (citing Nidhi Ravishankar, *Guillain-Barre Syndrome Following PCV Vaccine*, 2 CLINICS IN SURGERY 1 (2017) (Ex. 76); Hassan El Khatib et al., *Case Report: Guillain-Barre Syndrome with Pneumococcus – A New Association in Pediatrics*, 11 IDCASES 36 (2018) (Ex. 77)).) The first case report describes a 66-year-old female with a past medical history of hypertension, hyperlipidemia, and GERD, who developed GBS after receiving Prevnar 13 and the 23-valent pneumococcal conjugate ("Pneumovax 23") vaccinations. (Ravishankar, *supra*, at Ex. 76.) The second case report describes a 13-year-old male who presented with hypoactivity, slurred speech, and lower extremity weakness and developed septic shock due to pneumococcus with acute respiratory distress syndrome. (El Khatib et al., *supra*, at Ex. 77.) That patient was also found to have neurological findings of GBS. (*Id.*)

Dr. Steinman acknowledged two epidemiologic studies showing that pneumococcal vaccines do not pose an increased risk of GBS. (Ex. 22, p. 21 (citing Hung Fu Tseng et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults*, OPEN F. INFECTIOUS DISEASES, May 2018, at 1 (Ex. 45); Roger Baxter et al., *Lack of Association of Guillain-Barré Syndrome with Vaccinations*, 57 CLINICAL INFECTIOUS DISEASES 197 (2013) (Ex. 46; *see also* Ex. A, Tab 6).) However, he stressed that the study by Baxter et al., investigated results following administration of Pneumovax 23, not Prevnar 13. (*Id.* (citing Baxter et al., *supra*, at Ex. 46.) And while these epidemiologic studies are reassuring, Dr. Steinman contends they do not rule out Prevnar 13 as a cause of GBS. (*Id.*)

Regarding this particular case, Dr. Steinman observed that petitioner received the Prevnar 13 vaccine on January 15, 2015. (Ex. 22, p. 21 (citing Ex. 1, p. 1).) Twelve days later, she presented with a chief complaint of "ascending weakness," which he opined was the onset of GBS. (*Id.* (citing Ex. 10, pp. 80-81); Tr. 33-34.) Though there are no epidemiologic studies on point, he opined an interval of 12 days between immunization and GBS "certainly fits with the time course" reported by Schonberger et al. for GBS following swine flu immunization. (Ex. 22, p. 21 (citing Lawrence Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 100 AM. J. EPIDEMIOLOGY 105 (1979) (Ex. 47)); Tr. 41-42, 156-57.) Lastly, Dr. Steinman opined alternate causes, such as petitioner's type II diabetes, are highly unlikely to have caused her GBS. (Ex. 22, p. 22.)

b. Respondent's Experts

i. Vinay Chaudhry, M.D.[6]

Respondent filed two expert reports drafted by Dr. Chaudhry. (Exs. A, E.) Dr. Chaudhry opined that petitioner suffered from GBS that began with tingling on or about January 24, 2015 (nine days post-vaccination), peaked with quadriparesis on or about January 29, 2015, and improved to normal strength by March 8, 2016. (Ex. A, p. 5.) He further opined that petitioner suffered from the AIDP variant of GBS. (*Id.*) Dr. Chaudhry acknowledged that acute motor axonal neuropathy ("AMAN"), a variant of GBS, is known to be caused by the mechanism of molecular mimicry but disagreed that this can be extended to the AIDP form of GBS that petitioner suffered. (*Id.* at 7.) Dr. Chaudhry stressed, in particular, that "[i]t is well established that ganglioside antibodies arising as a result of molecular mimicry between the lipooligosaccharides of infective organisms and the surface molecules on the motor axons, induce axon injury by complement fixation, attracting macrophages and depositing membrane attack complexes in AMAN form of GBS." (Ex. E, p. 2 (citing Nobuhiro Yuki, *Ganglioside Mimicry and Peripheral Nerve Disease*, 35 MUSCLE & NERVE 691 (2007) (Ex. A, Tab 9); Peter D. Donofrio, *Guillain-Barré Syndrome*, 23 CONTINUUM (MINNEAPOLIS MINN.) 1295 (2017) (Ex. E, Tab 2); Francine J. Vriesendorp, *Guillain-Barré Syndrome: Pathogenesis*, UPTODATE, https://www.uptodate.com/contents/guillain-barre-syndrome-pathogenesis (last visited Nov. 18, 2020) (Ex. E, Tab 3)).) He stressed that gangliosides "have a completely different composition than phosphoglycerol moiety proposed by Dr. Steinman." (*Id.*) Unlike in AMAN, the specific antibody in AIDP has not been identified and its pathogenesis is not known, but Dr. Chaudhry suggested that it may be the result of a mechanism other than ganglioside mimicry. (*Id.*; Ex. A, p. 7 (citing Yuki, *supra*, at Ex. A, Tab 9).)

Dr. Chaudhry further observed that the authors of the Gilburd et al. paper concluded the autoantibodies produced by GBS patients to various phospholipids and nuclear antigens are likely "produced as a result of the myelin damage rather than cause the demyelination." (Ex. A, p. 8 (quoting Gilburd et al., *supra*, at Ex. 32).) He criticized Dr. Steinman's reinterpretation of the data from this paper and maintained "the authors did not find an association between the antibodies and GBS when compared to controls." (Ex. E, p. 2 (citing Gilburd et al., *supra*, at Ex. 32).) Dr. Chaudhry also contended the papers from Kanter et al., Ho et al., and Wang et al. discuss lipid arrays

[6] Dr. Chaudhry currently serves as a professor of neurology at Johns Hopkins University School of Medicine and co-director of the EMG Laboratory at Johns Hopkins Hospital. (Ex. A, p. 1.) He received his medical degree from the All India Institute of Medical Sciences, New Delhi, India. (Ex. B, p. 2.) He completed his clinical and research fellowships in neuromuscular diseases at Johns Hopkins University School of Medicine. (*Id.* at 3.) Dr. Chaudhry is board certified in Neurology, Neuromuscular diseases, Electrodiagnostic Medicine (Nerve Conduction and EMG), and Clinical Neurophysiology. (Ex. A, p. 1.) He holds an active clinical practice and sees close to 2000 patients a year (for over 25 years) mostly related to peripheral nerve disease. (*Id.*) He is involved in clinical research and has over 120 publications, including peer reviewed articles, reviews and book chapters. (*Id.*) Dr. Chaudhry has directly mentored over 50 fellows and 100s of medical students and neurology residents. (*Id.*) He routinely reviews articles for over 25 different journals and has previously served on the editorial board of three journals. (*Id.*)

in multiple sclerosis, which is a disease of the central nervous system in which oligodendrocytes, not Schwann cells, produce myelin. (Ex. A, p. 8 (citing Kanter et al., *supra*, at Ex. 33; Ho et al., *supra*, at Ex. 34; Wang et al., *supra*, at Ex. 35).) He stressed GBS and MS are two distinct diseases that do not share the same pathogenesis. (*Id.*) He further stressed that neither the subject vaccine nor the subject condition are discussed in these papers. (*Id.*) Importantly, the Nakos et al. manuscript also concludes "[i]t is not well understood whether these anti-phospholipid antibodies play a role in the pathogenesis of the polyneuropathy or represent a part of a more extensive immunoreaction that takes place in the GBS." (Ex. E, p. 2 (quoting Nakos et al., *supra*, at Ex. 55, p. 6).)

To demonstrate the safety of the Prevnar 13 vaccine, Dr. Chaudhry cited several studies monitoring post-vaccination adverse effects. (Ex. A, pp. 5-7.) Haber et al. evaluated the safety of Prevnar 13 by analyzing data from the Vaccine Adverse Event Reporting System ("VAERS") from June 2012 to December 2015. (*Id.* at 6 (citing Penina Haber et al., *Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged ⩾19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012-December 31, 2015*, 34 VACCINE 6330 (2016) (Ex. A Tab 5; *see also* Ex. 58)).) Dr. Chaudhry also cited Baxter et al., which included 13 years and 30 million person-years of data for a case-controlled analysis, finding no evidence of an increased risk of GBS following any vaccination or vaccinations combined. (*Id.* (citing Baxter et al., *supra*, at Ex. 46).) Although Prevnar 13 was not available for use during this study period, Dr. Chaudhry noted the 23-valent polysaccharide pneumococcal vaccine was included in the study. (*Id.*) Additionally, an article by Tseng et al., compared the safety of Prevnar 13 and Pneumovax 23 and found no evidence of increased risk of adverse events with Prevnar 13 among 313,136 doses of Prevnar 13. (*Id.* at 7 (discussing Tseng et al., *supra*, at Ex. 45).)[7] Jackson et al. studied the safety of Prevnar 13 among adults ≥ 70 years of age who were previously immunized with the Pneumovax 23 vaccine – finding no reported cases of GBS among the 936 vaccinated subjects. (*Id.* (citing Lisa Jackson et al., *Immunogenicity and Safety of a 13-Valent Pneumococcal Conjugate Vaccine in Adults 70 Years of Age and Older Previously Vaccinated with 23-Valent Pneumococcal Polysaccharide Vaccine,* 31 VACCINE 3585 (2013) (Ex. A Tab 7)).) Lastly, Dr. Chaudhry noted that seven clinical studies conducted in the U.S. and Europe assessed the safety of Prevnar 13 and included 91,593 adults (48,806 of which received Prevnar 13) ranging in age from 18 through 101 years. (*Id.* (citing *Pfizer Medical Information: Prevnar 13*, PFIZER, https://www.pfizermedicalinformation.com/en-us/prevnar-13 (last visited Oct. 19, 2019) (Ex. A, Tab 8, pp. 8-12)).) Dr. Chaudhry observed that serious adverse events were reported within one month of vaccination in 0.2%-1.4% of 5,057 subjects vaccinated with Prevnar 13, but GBS was not among the reported events. *(Id.*)

Dr. Chaudhry contended that, despite Dr. Steinman's great lengths to identify phosphoglycerol in Prevnar 13, he "could never conclusively ascertain if phosphoglycerol is present in Prevnar 13," and asserted the conformational structure

[7] In his report, Dr. Chaudhry miscited this study as "Vaccine 2013," which would seem to refer to Jackson et al., *infra*.

between an exogenous agent and self-antigen alone are not sufficient to prove that molecular mimicry is the pathogenic mechanism for a disease. (Ex. E, pp. 2-3.) Rather, Dr. Chaudhry offered four criteria he opines must be satisfied to conclude that a disease such as GBS is triggered by molecular mimicry: (1) an epidemiological association between the infectious agent and the immune-mediated disease, (2) identification of T cells or antibodies directed against the patient's target antigens, (3) identification of microbial mimics of the target antigen, and (4) reproduction of the disease in an animal model. (*Id.* at 3 (citing Yuki, *supra*, at Ex. A, Tab 9).) According to Dr. Chaudhry, none of these criteria have been met for Prevnar 13. (*Id.*)

Lastly, Dr. Chaudhry disputed petitioner's reliance on the time frame for GBS following swine flu vaccination, stressing that temporal association in one case does not support causation and that none of the Prevnar 13 studies reported GBS in pre-licensure and post-licensure cases up to 42 days post-vaccination. (Ex. A, p. 9.)

ii. Robert S. Fujinami, Ph.D.[8]

Dr. Fujinami filed four expert reports on behalf of respondent. (Exs. C, F-H.) He also provided testimony at the entitlement hearing. (Tr. 168.) Dr. Fujinami opined that, if Dr. Steinman's theory were correct, then the Prevnar 13 vaccine "would induce pathogenic antibodies against phosphatidylcholine, which is contained in all myelin." (Ex. C, p. 3.) Because myelin is present in both the peripheral nervous system and the central nervous system, Dr. Fujinami explained that the phospholipids that are central to Dr. Steinman's theory are thus contained in both the peripheral nervous system and the central nervous system; however, GBS is an immune mediated disease of the peripheral nervous system, not the central nervous system. (*Id.*) He questioned, then, why the neuroinflammation putatively caused by antibodies to phosphatidylcholine only induces damage in the peripheral nervous system and not the central nervous system. (*Id.*) To that end, in this case, petitioner did not experience central nervous system involvement. (*Id.*) Petitioner's CT and MRI of her head, as well as blood work, were all normal. (*Id.* (citing Ex. 22, p. 5).) If there was central nervous system involvement with inflammation in the myelin, Dr. Fujinami stressed it would be noted in the medical records. (*Id.*)

[8] Dr. Fujinami currently serves as a professor in the Department of Pathology, Division of Microbiology and Immunology, and an Adjunct Professor in the Department of Neurology at the University of Utah School of Medicine. (Ex. D, p. 1; Tr. 168.) He received his undergraduate degree in microbiology from the University of Utah and doctorate from Northwestern University. (Ex. D, p. 1; Tr. 169.) Dr. Fujinami completed his postdoctoral training at The Scripps Research Institute and advanced to Assistant Professor investigating how viruses/infections could induce autoimmune disease. (Ex. D, p. 1; Tr. 169-70.) While at Scripps, Dr. Fujinami and Dr. Michael Oldstone first introduced molecular mimicry as a theoretical concept in the early 1980's, with respect to viruses and their putative role in the propagation of autoimmunity. (Ex. C, p. 1.) He is accordingly one of the scientific "fathers" of the concept, though he is not a medical doctor. Dr. Fujinami has published hundreds of articles about microbial infection, vaccines, and autoimmune disease. (Ex. D, pp. 20-37.) He has also served on various panels for the National Institute of Health, Institute of Medicine, National Academy of Sciences, National Science Foundation, and National Multiple Sclerosis Society. (Ex. C, p. 2.) At the hearing, Dr. Fujinami was proffered as an expert in immunology, neuroimmunology, and neurobiology without objection. (Tr. 183.)

Like Dr. Chaudhry, Dr. Fujinami is critical of Dr. Steinman's reliance on the Gilburd et al. study. (Ex. C, p. 3.) He too stressed that Gilburd et al. concluded the autoantibodies to various phospholipid antigens were probably produced as a result of myelin damage, rather than the cause of demyelination. (*Id.*; Tr. 186-87.) However, he recognized that the authors did not specifically look at whether the phospholipid antigens caused demyelination. (Tr. 259-63.)

Dr. Fujinami also took issue with Dr. Steinman's reinterpretation of the Ho et al. paper. (Ex. G, pp. 1-2 (citing Ho et al., *supra*, at Ex. 34); Tr. 208-09.) Dr. Fujinami stressed that Ho et al. did not address "what initiated the antibody response or the extensive inflammation" in the central nervous system of MS patients. (Ex. G, p. 2 (citing Ho et al. *supra*, at Ex. 34).) He also disagreed with Dr. Steinman's emphasis on the polar head group containing a phosphate molecule as demonstrated in the Ho et al. article. (Tr. 209-10.) He explained his understanding of Dr. Steinman's opinion regarding the affinity of the antibody binding to the glycolipid as depending on the length of the carbon chains that are hooked to the phosphate group; however, Dr. Fujinami pointed out that these phosphate head groups appear in both the non-reactive, as well as the reactive, entities. (*Id.* at 209-10, 229-30.) He thus interpreted the Ho et al. article as demonstrating that "not all molecules that have that phosphate group react with the antibodies." (*Id.* at 230.) In fact, with regard to all of the examples provided by Dr. Steinman (Exs. 32, 34, 55), Dr. Fujinami opined the authors are investigating the nature of the autoantibodies, and "not the induction of the autoantibodies. They do not speak to phospholipids inducing neuroinflammatory disease." (Ex. G, p. 2 (citing Gilburd et al., *supra*, at Ex. 32; Ho et al., *supra*, at Ex. 34; Nakos et al., *supra*, at Ex. 55); Tr. 224, 226-27, 280-83.)

Contrary to Dr. Steinman's analysis, Dr. Fujinami opined that if phosphatidylcholine, or derivatives of this structure, are present in the Prevnar 13 vaccine (19A component), then based on the findings by Ho et al., the structure should protect against, rather than enhance or cause, neuroinflammation. (Ex. C, p. 3 (citing Ho et al., *supra*, at Ex. 34); Tr. 208-09.) Dr. Steinman and his colleagues in Ho et al. treated mice immunized with a peptide derived from myelin (in powerful adjuvants) with a derivative of oxidized phosphatidylcholine (PGPC) using different treatment regimens. (Ex. C, p. 4 (citing Ho et al., *supra*, at Ex. 34).) Yet, Dr Fujinami observed in all of the different treatments the disease was markedly reduced. (*Id.*) Specifically, the authors observed that "PGPC unexpectedly reduced the severity of EAE throughout the disease course." (*Id.* (quoting Ho et al., *supra*, at Ex. 34, p. 3).)[9] The authors found that PGPC suppressed autoreactive T cell activation. (*Id.*) The authors even go so far as to conclude "[t]he identification of lipids targeted by autoantibodies affords the opportunity to mine small lipid-soluble molecules as potential new drugs for autoimmune disease." (*Id.* (quoting Ho et al., *supra*, at Ex. 34, p. 10).) According to Dr. Fujinami, Dr. Steinman is opining that phosphatidylcholine in the vaccine causes GBS, while in the Ho et al. paper (which he is a co-author of), he concludes the lipids like phosphatidylcholine could prevent or treat autoimmune disease. (*Id.*)

[9] "PGPC" is 1-palmitoyl-2-glutaroyl-sn-glycero-3-phosphocholine. (Ho et al., *supra*, at Ex. 34, p. 2.)

Dr. Fujinami noted, as Dr. Steinman does, that the Prevnar 13 vaccine contains aluminum phosphate adjuvant (alum). (Ex. C, p. 4.) In response to petitioner's theory, Dr. Fujinami presented work from two groups independently published in peer-reviewed journals showing that genetically susceptible animals immunized with whole myelin, or myelin derived proteins, adsorbed to alum did not develop autoimmune neuroinflammatory disease (*i.e.*, EAE, a preclinical model for MS). (*Id.* (citing Maryline Sicotte et al., *Immunization with Myelin or Recombinant Nogo-66/MAG in Alum Promotes Axon Regeneration and Sprouting After Corticospinal Tract Lesions in the Spinal Cord*, 23 MOLECULAR & CELLULAR NEUROSCIENCE 251 (2003) (Ex. C, Tab 2); Maja Wållberg et al., *Vaccination with Myelin Oligodendrocyte Glycoprotein Adsorbed to Alum Effectively Protects DBA/1 Mice from Experimental Autoimmune Encephalomyelitis*, 33 EUR. J. IMMUNOLOGY 1539 (2003) (Ex. C, Tab 3)); Tr. 241-43.) Wållberg et al. also demonstrated that "genetically susceptible mice 'vaccinated' with myelin oligodendrocyte glycoprotein adsorbed to alum were actually protected from the autoimmune neuroinflammatory disease." (Ex. C, p. 4 (citing Wållberg et al., *supra*, at Ex. C, Tab 3); Tr. 242-43.) Further, Dr. Fujinami explained that Sicotte et al. observed that myelin/alum immunized animals were found to have better axon repair when the spinal cord was subsequently damaged. (Ex. C, p. 4 (citing Sicotte et al., *supra*, at Ex. C, Tab 2); Tr. 241-42.) Thus, Dr. Fujinami contended that myelin adsorbed to alum (like the glycoconjugates in the formulation of the Prevnar 13 vaccine) injected into experimental animals does not induce EAE and that this immunization can protect against subsequent attempts to induce EAE. (Ex. C, p. 4 (citing Prevnar 13 Package Insert, *supra*, at Ex. 31, p. 25); Tr. 243-45.) While Dr. Steinman opined that various cytokines, including IL-18, are found in GBS patients, Dr. Fujinami indicated "there is no link showing that an alum-based vaccine is able to persist and drive a chronic IL-18 response in autoimmune disease." (Ex. F, p. 2.)

Dr. Fujinami further suggested that Dr. Steinman erroneously discounted the Tseng et al. and Baxter et al. studies because they focus on Pneumovax 23 and not Prevnar 13. (Ex. C, p. 5.) Dr. Fujinami explained that the polysaccharide vaccine contains the same 19A component of pneumococcus as the conjugate vaccine. (*Id.* (citing Usama Assaad et al., *Pneumonia Immunization in Older Adults: Review of Vaccine Effectiveness and Strategies*, 7 CLINICAL INTERVENTIONS AGING 453 (2012) (Ex. C, Tab 1)).) Thus, if the 19A component is of importance to petitioner's theory, Dr. Fujinami stressed it is contained in both pneumococcal vaccines. (*Id.*) Dr. Fujinami also responded to Dr. Steinman's opinion that putative pathogenic phospholipids are contained in the *Streptococcus pneumoniae* ("*S. pneumoniae*") strains used in the formulation of the vaccine. (Ex. F, p. 1.) Petitioner theorizes that polysaccharides and phospholipids, present in the strains of *S. pneumonia*, generate the putative pathogenic antibodies involved with GBS. (*Id.*) Following this logic, Dr. Fujinami suggested that if the same elements (polysaccharides and phospholipids) are present in the bacteria, individuals infected with *S. pneumoniae* should also develop GBS. (*Id.*; Tr. 217-19.) However, he stressed this is not the case. (Ex. F, p. 1; Tr. 217-19.) He acknowledged that two case reports apparently link *S. pneumoniae* and GBS, though he opined these reports provide little to no support for Dr. Steinman's theory. (Ex. F, pp. 1-2; Tr. 218.)

V. Discussion

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," showing that the subject vaccine can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004)). Such a theory need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *See Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [the proposed causal] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Boatmon*, 941 F.3d at 1359 (first quoting *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010); then quoting *Knudsen*, 35 F.3d at 548-49).

As with many cases in the Program, petitioner's theory involves molecular mimicry. (Ex. 22, p. 10.) Molecular mimicry is a concept with several constituent parts whereby a susceptible host encounters a foreign antigen that has sufficient similarity ("homology") with components of host tissue such that the immune system "cross-reacts," producing antibodies that attack the host tissue instead of the foreign antigen to ultimately cause disease or injury. (Tr. 44-45; INST. OF MED., ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al. eds., 2012) [hereinafter IOM Report] (Ex. 71, p. 99).) Molecular mimicry "is a generally accepted scientific principle, [but] mere invocation of the scientific term does not carry a petitioner's burden in a Program case." *Deshler v. Sec'y of Health & Human Servs*., No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec'y of Health & Human Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019)). "[T]he finding of sequence homology does not necessarily mean the similarity has significance to the immune system." *Tullio v. Sec'y of Health & Human Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *aff'd*, 149 Fed. Cl. 448 (2020); *see also Caredio ex rel. D.C. v. Sec'y of Health & Human Servs.*, No. 17-0079V, 2021 WL 4100294, at *31 (Fed. Cl. Spec. Mstr. July 30, 2021) ("[D]emonstration of homology alone is not enough to establish a preponderant causation theory." (emphasis omitted) (citing *Schultz v. Sec'y of Health & Human Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020))), *mot. for rev. denied*, No. 17-79V, 2021 WL 6058835 (Fed. Cl. Dec. 3, 2021).

In that regard, Dr. Chaudhry asserts on respondent's behalf that four criteria – (1) supportive epidemiology, (2) identification of T cells or antibodies directly against human antigens, (3) identification of the mimics of the target antigen, and (4) reproduction in an

animal model – must be demonstrated in order to conclude that GBS can be triggered by molecular mimicry following Prevnar 13 vaccination. (Ex. E, p. 3 (citing Yuki, *supra*, at Ex. A, Tab 9).) As noted above, however, petitioners in this Program are not required to establish scientific certainty. *See, e.g.*, *Gross v. Sec'y of Health & Human Servs.*, No. 17-1075V, 2022 WL 9669651, at *36 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (finding that criteria such as proposed by Dr. Chaudhry is tantamount to "require[ing] scientific certainty, which is a bar too high"). With regard to the application of molecular mimicry, prior cases have expressed that "[t]he line must be drawn somewhere between speculation and certainty." *Brayboy v. Sec'y of Health & Human Servs.*, No. 15-183V, 2021 WL 4453146, at *19 (Fed. Cl. Spec. Mstr. Aug. 30, 2021). Thus, for example, in *Brayboy*, an omnibus proceeding addressing autoimmune premature ovarian insufficiency, the special master found it sufficient that the petitioners "identified cross-reaction between components of the vaccine and proteins in the body that are directly responsible for the health and productivity of the organ at issue" and further expressed that requiring additional steps, or insisting on direct, testable evidence, would impermissibly heighten petitioners' burden of proof. *Id.*

GBS is an acute inflammatory polyneuropathy affecting the peripheral nerves and, in the case of AIDP, most notably affecting myelin tissue. (Hugh J. Willison et al., *Guillain-Barré Syndrome*, 388 LANCET 717 (2016) (Ex. A, Tab 1, pp. 1, 3).) While the condition is believed to be of an autoimmune etiology, the pathogenesis of GBS is incompletely understood. (Nakos et al., *supra*, at Ex. 55; Gilburd et al., *supra*, at Ex. 32, p. 2; Willison et al., *supra*, at Ex. A, Tab 1; Tr. 76.) Although there is no established association between *S. pneumoniae* infection and GBS (Ex. A, p. 6; Tr. 217), it is generally accepted that a number of different infectious antigens can cause GBS, including unspecified upper respiratory infections (*Guillain-Barré Syndrome Information Page*, NAT'L INST. NEUROLOGICAL DISORDERS & STROKE, https://www.ninds.nih.gov/Disorders/All-Disorders/Guillain-Barré-Syndrome-Information-Page (last visited Jul. 25, 2017) (Ex. 29); Baxter et al., *supra*, at Ex. 46, pp. 1, 4; Willison et al., *supra*, at Ex. A, Tab 1, pp. 2, 4). This includes both viral and bacterial infections. (Willison et al., *supra*, at Ex. A, Tab 1, p. 4; Baxter et al., *supra*, at Ex. 46, p. 1.) Additionally, for at least one of these antigens, *C. jejuni*, there is sufficient proof to conclude that molecular mimicry is the mechanism of causation leading to GBS, albeit resulting primarily in the axonal subtype of GBS and involving a molecular mimic not at issue here. (Ex. A, p. 5; Ex. E, pp. 1,3; Tr. 76, 161; Yuki, *supra*, at Ex. A, Tab 9, p. 2; IOM Report, *supra*, at Ex. 71, pp. 100-01.) Evidence also supports homology between various other antigens and myelin tissue. (*See* IOM Report, *supra*, at Ex. 71, p. 100 (hepatitis B virus); Willison et al., *supra*, at Ex. A, Tab 1, p. 2 (noting cases of GBS shortly after rabies vaccination).) Thus, there is little question that multiple antigens are implicated as causes of GBS. Additionally, Dr. Chaudhry agrees on respondent's behalf that at least some formulations of the flu vaccine have been identified as a cause of GBS, suggesting that antigenic triggers for GBS are not limited to active infections. (Ex. A, p. 6.) Petitioner's expert opines that this understanding of GBS provides some basic support for his opinion that the Prevnar 13 vaccine can cause GBS. (*See* Tr. 76; Ex. 75, p. 13.)

In some prior cases, this background information has partly informed the special masters' analysis of a petitioner's theory of causation with respect to GBS. *See, e.g.*, *J.G. v. Sec'y of Health & Human Servs.*, No. 20-664V, 2023 WL 2752634, at *30 (Fed. Cl. Spec. Mstr. Feb. 13, 2023) (observing that "[t]he experts do not dispute the theory of molecular mimicry, or that it is a sound and reliable theory generally as it relates to GBS" and that "[m]olecular mimicry has been accepted as a sound and reliable theory in many Vaccine Program cases dealing with demyelinating conditions, including GBS"); *Osso v. Sec'y of Health & Human Servs.*, No. 18-575V, 2023 WL 5016473, at *21 (Fed. Cl. Spec. Mstr. July 13, 2023) (molecular mimicry accepted as a "sound and reliable theory"); *Harris v. Sec'y of Health & Human Servs.*, No. 18-944V, 2023 WL 2583393, at *22 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (finding that "the fact that GBS is well accepted as an autoimmune condition with a wide variety of suspected antigenic triggers, inclusive of antigens from both infection and vaccination, provides meaningful evidence supporting petitioner's burden of proof with respect to *Althen* prong one"). *But see Trollinger v. Sec'y of Health & Human Servs.*, No. 16-473V, 2023 WL 2521912, at *30 (Fed. Cl. Spec. Mstr. Feb. 17, 2023) (finding that "Dr. Steinman's theory had a one-size-fits-all quality, in which he strained to shoehorn the science behind the flu-GBS association into the context of the pneumococcal vaccine" and further noting that, "[i]f this were sufficient to establish that this particular vaccine 'can cause' GBS, it is hard to imagine the theory not also applying to *each and every one* of the sixteen Program-covered vaccines/vaccine antigenic components"), *mot. for rev. denied*, 167 Fed. Cl. 127 (2023). Although only the flu vaccine is presumed to be a cause of GBS in this Program (42 C.F.R. § 100.3(a)), petitioners have been found entitled to compensation in at least isolated instances for GBS caused by many other vaccines. This includes vaccines that target both viruses and bacteria. *See, e.g.*, *Salmins v. Sec'y of Health & Human Servs.*, No. 11-140V, 2014 WL 1569478, at *14 (Fed. Cl. Spec. Mstr. Mar. 31, 2014) (finding that HPV vaccine "can cause" GBS); *Peugh v. Sec'y of Health & Human Servs.*, No. 99-638V, 2007 WL 1531666, at *17 (Fed. Cl. Spec. Mstr. May 8, 2007) (finding as part of an omnibus proceeding that hepatitis B vaccine can GBS); *Whitener v. Sec'y of Health & Human Servs.*, No. 06-0477V, 2009 WL 3007380, at *20 (Fed. Cl. Spec. Mstr. Sept. 2, 2009) (finding that meningococcal vaccine can cause GBS); *Koller v. Sec'y of Health & Human Servs.*, No. 16-439V, 2021 WL 5027947, at *7-20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021) (finding that pneumococcal conjugate vaccine, Prevnar 13, can cause GBS); *Mohamad v. Sec'y of Health & Human Servs*., No. 16-1075V, 2022 WL 711604, at *9-18 (Fed. Cl. Spec. Mstr. Jan. 27, 2022) (finding that Tdap vaccine can cause GBS); *J.G.*, 2023 WL 2752634, at *29-32 (finding that hepatitis A vaccine can cause GBS). In fact, given the nature of the condition, molecular mimicry has been accepted as a theory of causation for GBS even in the absence of *any* demonstration of homology and cross-reaction. *Salmins*, 2014 WL 1569478, at *14.

Within that context, Dr. Steinman opines that the Prevnar 13 vaccine can be considered among the causes of GBS, presenting several pieces of medical literature to demonstrate that (1) the Prevnar 13 vaccine contains phosphoglycerol groups that are necessary to the vaccine's immunogenicity (Ex. 50, pp. 18-19 (citing Chang et al., *supra*, at Ex. 52; Prevnar 13 Patent, *supra*, at Ex. 51); Tr. 96 (citing Bryson et al., *supra*, at Ex. 103)); (2) the phosphate portion of the phospholipid molecule has immune

reactivity in myelin tissue, albeit demonstrated in the context of a different demyelinating condition (multiple sclerosis) (Ex. 22, p. 13 (quoting Ho et al., *supra*, at Ex. 34); Ex. 50, pp. 8, 12 (citing Ho et al., *supra*, at Ex. 34); Tr. 94-95); (3) GBS patients develop anti-phospholipid antibodies (Ex. 22, pp. 13-14 (citing Gilburd et al., *supra*, at Ex. 32); Ex. 50, pp. 11-12 (citing Nakos et al., *supra*, at Ex. 55)), and (4) these antibodies are cross-reactive with phospholipids present in myelin tissue (Ex. 50, p. 4 (citing Gilburd et al., *supra*, at Ex. 32; Ho et al., *supra*, at Ex. 34); Tr. 94-95).

Dr. Steinman's theory does not involve the anti-ganglioside antibodies that are most commonly associated with GBS. However, literature filed in this case indicates that fewer than half of GBS patients have anti-ganglioside antibodies. (Donofrio, *supra*, at Ex. E, Tab 2, pp. 1-2.) Thus, we do not actually know the full scope of the antibodies that may be implicated in the pathology of GBS, leaving little reason to doubt Dr. Steinman's theory on that basis. (*See, e.g.*, *id.* at 11 ("Since a wide range of viruses and bacterial agents can incite an antibody in AIDP, it has been difficult to find a common antigenic stimulus for the illness. Equally difficult has been the identification of specific antibody biomarkers in myelin."); Willison et al., *supra*, at Ex. A, Tab 1, p. 1 (noting that "there are many clinical and investigative components to consider" when diagnosing GBS, "especially in atypical cases," and that "diagnostic biomarkers are not available for most variants of the syndrome" as "[i]dentification of biomarkers and establishment of their pathophysiological roles, if any, in experimental models has been a major research challenge"); *accord Gross*, 2022 WL 9669651, at *36 (indicating that "the literature filed by the parties does not support the notion that gangliosides are the only player in the game of molecular mimicry").) To this point, I have previously emphasized that the "S" in GBS stands for "syndrome" and that "GBS variants are generally believed to have a multitude of both clinical presentations and causes," *McGill v. Sec'y of Health & Human Servs.*, No. 15-1485V, 2023 WL 3813524, at *27 n.16 (Fed. Cl. Spec. Mstr. May 11, 2023), raising the question of whether a single causal theory can explain every instance of GBS, post-vaccination or otherwise.

I have previously concluded that Dr. Steinman's phosphoglycerol theory is sound and reliable, and preponderantly supports a legally probable, though not scientifically certain, theory of causation sufficient to satisfy petitioner's burden of proof under *Althen* prong one, based on the same medical literature cited in this case. *Pierson v. Sec'y of Health & Human Servs.*, No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Bartoszek v. Sec'y of Health & Human Servs.*, No. 17-1254V, 2024 WL 4263604, at *17-22 (Fed. Cl. Spec. Mstr. Aug. 27, 2024); *see also Cooper v. Sec'y of Health & Human Servs.*, No. 18-1885V, 2024 WL 1522331, at *13-18 (Fed. Cl. Spec. Mstr. Mar. 12, 2024). I again reach the same conclusion based on the evidence of record in this case. Additionally, other special masters have reached similar conclusions based on substantially similar underlying evidence. *Koller*, 2021 WL 5027947, at *16-20 (Gowen); *Maloney v. Sec'y of Health & Human Servs*., No. 19-1713V, 2022 WL 1074087, at *30-31 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (Dorsey); *Gross*, 2022 WL 9669651, at *35-36 (Dorsey); *Sprenger v. Sec'y of Health & Human Servs*., No. 18-279V, 2023 WL 8543435, at *18-19 (Fed. Cl. Spec. Mstr. Nov. 14, 2023) (Dorsey); *Parker v. Sec'y of Health & Human Servs.*, No. 20-411V, 2023 WL 9261248,

at *20-22 (Fed. Cl. Spec. Mstr. Dec. 20, 2023) (Dorsey); *Anderson v. Sec'y of Health & Human Servs.*, No. 18-484V, 2024 WL 557052, at *30-31 (Fed. Cl. Spec. Mstr. Jan. 17, 2024) (Dorsey); *Simeneta v. Sec'y of Health & Human Servs.*, No. 18-859V, 2024 WL 4881411, at *30-33 (Fed. Cl. Spec. Mstr. Oct. 31, 2024) (Dorsey); *see also Tracy ex rel. R.S. v. Sec'y of Health & Human Servs*., No. 16-213V, 2022 WL 1125281, at *29-32 (Fed. Cl. Spec. Mstr. Mar. 30, 2022) (Special Master Sanders accepting a similar theory in the context of transverse myelitis).[10]

However, acceptance of this theory has not been unanimous among special masters. *Bielak v. Sec'y of Health & Human Servs.*, No. 18-761V, 2023 WL 35509, at *33-37 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (Corcoran); *Trollinger*, 2023 WL 2521912, at *26-30 (Corcoran); *Gamboa-Avila v. Sec'y of Health & Human Servs*., No. 18-925V, 2023 WL 6536207, at *25-29 (Fed. Cl. Spec. Mstr. Sept. 11, 2023) (Corcoran), *mot. for rev. denied*, 170 Fed. Cl. 441 (2024), *appeal filed*, No. 24-1765 (Fed. Cir. May 1, 2024); *Jaye v. Sec'y of Health & Human Servs.*, No. 20-672V, 2024 WL 3691413, at *14-17 (Fed. Cl. Spec. Mstr. July 18, 2024) (Corcoran); *Morrison v. Sec'y of Health & Human Servs.*, No. 18-386V, 2024 WL 3738934, at *19-21 (Fed. Cl. Spec. Mstr. July 18, 2024) (Oler).[11] These contrary decisions are not binding on me. *Boatmon*, 941 F.3d at 1358-59; *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). Nonetheless, I have considered the points raised by these decisions. I simply reach a different conclusion based on my overall weighing of the evidence on this record. Notably, even when reaching a different result, there has still been agreement that record evidence comparable to what has been presented in this case at a minimum

> *does* offer reliable support for the conclusion that phosphoglycerol is found in the pneumococcal vaccine; that the immune system produces antibodies in reaction to the relevant antigens containing the phosphoglycerol; and that individuals with neuropathies (although some suffer from the distinguishable disease MS) have been shown in small sample studies to possess antibodies specific to myelin-containing phospholipids.

Trollinger, 2023 WL 2521912, at *28.

[10] Other special masters have also found that petitioners have preponderantly established that the Prevnar 13 vaccine case cause GBS based on the other causal theory Dr. Steinman presented. *Byrd v. Sec'y of Health & Human Servs.*, No. 20-1476V, slip op. (Fed. Cl. Spec. Mstr. July 8, 2024) (Gowen) (accepting petitioner's causal theory based on molecular mimicry between CRM197 and contactin-1); *Anderson*, 2024 WL 557052, at *31-32 (Dorsey) (same); *Sprenger*, 2023 WL 8543435, at *19-20 (Dorsey) (same); *Gross*, 2022 WL 9669651, at *36-37 (Dorsey) (same); *Maloney*, 2022 WL 1074087, at *32 (Dorsey) (same).

[11] Some additional cases were resolved against petitioners based on different theories of causation. *McConnell v. Sec'y of Health & Human Servs.*, No. 18-1051V, 2022 WL 4008238, at *7-9 (Fed. Cl. Spec. Mstr. Aug. 19, 2022); *Deshler*, 2020 WL 4593162, at *19-21.

I have considered the issues raised by respondent's experts in this case, but do not find that they cast sufficient doubt on Dr. Steinman's theory such that they would prevent petitioner from meeting her burden of proof under *Althen* prong one.[12]

As a threshold matter, respondent's experts stress that the *S. pneumoniae* has carbohydrates in common with the vaccine and contend that the fact that there has been no recognized causal association between the pneumococcal infection and GBS undercuts any causal role for phosphoglycerol in the Prevnar 13 vaccine. (Tr. 218-19; Ex. F, pp. 1-2; Ex. E, p. 1.) However, Dr. Fujinami explained during the hearing that he cannot say with certainty whether the specific phosphoglycerol component of the vaccine that Dr. Steinman relies upon is present in the natural bacterium. (Tr. 291.) In any event, petitioner need not "demonstrate that a vaccine's infectious counterpart is a known-disease trigger." *Morrison*, 2024 WL 3738934, at *18.

Additionally, respondent stresses three epidemiologic studies that he contends should weigh against any theory that the Prevnar 13 vaccine can cause GBS. (ECF No. 135, pp. 17-18.) The Federal Circuit has previously stressed that a petitioner is not obligated to prove a case with epidemiology. *Capizzano*, 440 F.3d at 1325. Yet, "[n]othing in *Althen* or *Capizzano* requires the Special Master to ignore probative epidemiological evidence that undermines petitioner's theory." *D'Tiole v. Sec'y of Health & Human Servs.*, 726 F. App'x 809, 811 (Fed. Cir. 2018). Baxter et al. is a 2013 study that examined 415 incident cases of GBS from the Kaiser Permanente Northern California ("KPNC") patient population to compare "the odds of vaccination in the 6 and 10 weeks prior to onset of GBS to the odds of vaccination during the same time intervals in all vaccinated individuals in the entire KPNC population." (Baxter et al., *supra*, at Ex. A, Tab 6, p. 1.) The study included two instances of GBS occurring within 6 weeks of administration of the polysaccharide pneumococcal (as opposed to conjugate) vaccine. (*Id*. at 6 tbl.2.) The study did not detect any increased risk of GBS following any vaccination, even flu vaccination. (*Id*. at 5-6.) However, despite the large size of the study, the authors concluded that "we are unable to exclude any possible association between vaccines and GBS," explaining that "we had limited power to fully assess the risk of GBS following vaccination due to the rarity of the outcome." (*Id*. at 7.) Haber et al. is a 2016 study that used Vaccine Adverse Events Reports System ("VAERS") data to examine the safety profile of the Prevnar 13 vaccine. (Haber et al.,

[12] There have also been some significant differences in the record evidence of the various decisions weighing the theory Dr. Steinman presents in this case. For instance, while some prior cases denying compensation for GBS based on this theory found significance in the distinction between B- and T-cell responses, respondent's expert in *Cooper* specifically disclaimed a hardline distinction between such responses, testifying instead that "nothing is pure B cell/Tcell". *Compare Cooper*, 2024 WL 1522331, at *17, *with Deshler*, 2020 WL4593162, at *19-20, *and Bielak*, 2023 WL 35509, at *29-37. The *Morrison* decision turned in part on acceptance of the pathological role of anti-ganglioside antibodies in GBS as suggested by Dr. Whitton. 2024 WL 3738934, at 19-21. Yet, respondent's expert in *Bartoszek*, Dr. Kedl, asserted to the contrary that this is "tenuous, speculative and clinically unproven," and that "the vast majority of literature stands in sharp contrast to the assertion that there is any causal relationship between ganglioside-specific antibodies and GBS." 2024 WL 4263604, at *19-20, n.16. Thus, despite the accumulating number of decision and rulings by special masters, it is still important to look closely at respondent's expert presentation in each case. (ECF No. 135, p. 24, n.17 (respondent urging a different result from the undersigned's prior *Pierson* case based on the rebuttal evidence filed in this case).)

supra, at Ex. A, Tab 5.) Eleven cases of GBS were reported within 42 days of a Prevnar 13 vaccination, which would constitute 0.7 cases per million doses of the vaccine as distributed. (*Id*. at 5.) However, the authors cautioned that VAERS is a passive surveillance system, meaning that the data is susceptible to underreporting, specific reports are of varying quality, and no control group is available. (*Id*.) Finally, in 2018, Tseng et al. compared adverse events among the elderly following the polysaccharide pneumococcal vaccine against adverse events following the conjugate pneumococcal vaccine. (Tseng et al., *supra*, at Ex. 45.) Four instances of GBS were observed following the conjugate vaccine as compared to eight instances of GBS following the polysaccharide. (*Id.* at 6 tbl.3.) Therefore, the authors concluded that the risk of GBS following the conjugate vaccine was no greater than any risk following the polysaccharide vaccine. (*Id*. at 7.) However, the study did not assess the observed rate of GBS following the polysaccharide against any control, though the authors alluded to the polysaccharide vaccine as having a "well-established" safety profile. (*Id.*) I have considered all the epidemiology submitted in this case, and although I give some weight to these studies highlighted by respondent, I do not find that they cast significant doubt on the viability of Dr. Steinman's theory.

Regarding the details of Dr. Steinman's theory, respondent's experts also contend that, although there is evidence in the medical literature that phospholipid antibodies may enhance disease after disease onset, there is no evidence to support a causal role for these antibodies. (Tr. 187-90, 194-95, 232; Ex. A, p. 8; Ex. E, p. 2.) They specifically note that Gilburd et al. suggested that the autoantibodies found in GBS patients were "a result of myelin damage, rather than cause the inflammatory demyelination." (Ex. A, p. 8 (quoting Gilburd et al., *supra*, at Ex. 32); Tr. 187-90, 262-63.) Dr. Fujinami further hypothesized that the antibodies are better explained by epitope spreading. (Tr. 219-32.) However, Dr. Steinman observed that there is no evidence in the literature to support the assertion that these antibodies are merely an after effect of demyelination, noting that they "come up very early" in GBS patients that were tested. (*Id.* at 292, 295-96.) He further noted that Nakos et al. examined "real disease" and found that seven out of nine GBS patients had relevant antibodies within days. (*Id.* at 312, 315-17.) According to Dr. Steinman, the timeframe within which the Nakos et al. study detected the antibodies leaves epitope spreading "the least likely" explanation for the data. (*Id.* at 322; *accord Greenslade v. Sec'y of Health & Human Servs*., No. 14-1140V, 2024 WL 3527665, at *38 (Fed. Cl. Spec. Mstr. June 28, 2024) (crediting expert testimony that epitope spreading takes "a few weeks" and develops after significant tissue damage has already occurred).)

Dr. Fujinami also asserted that Dr. Steinman placed too much emphasis on the Ho et al. study and, more specifically, on any causal role for the phosphate head group. (Tr. 209, 230 (citing Ho et al., *supra*, at Ex. 34).) He noted that both the reactive and non-reactive molecules identified by Ho et al. contain a phosphate head group and have a similar structure. (*Id.* at 209-10, 229-30.) He thus opined the literature demonstrates that "not all molecules that have that phosphate [head] group react with the antibodies." (*Id.* at 230.) Dr. Steinman agreed with Dr. Fujinami's assessment of the Ho et al. study as demonstrating that not all antibodies will bind to phosphoglycerol but explained that

this is due to the fact that the antibodies that recognize phosphoglycerol also recognize what is attached. (*Id.* at 304-08.) He noted that there was also evidence in the Ho et al. study that the lipid side chains, which include the phosphoglycerol head group, actually facilitate antibody binding. (*Id.* at 306.) However, he explained that Barbar et al. showed that "no matter what's attached to those phosphate groups, those antibodies are still going to be recognizing the phosphate head group." (*Id.* at 128-30, 136; Ex. 96, p. 5 (citing Barbar et al., *supra*, at Ex. 102).) Thus, he explained that while there are "exceptions to the set of observation we made" in the Ho et al. study, there is "a confluence of evidence from this paper [Ho et al.], from Bryson, from Nakos, even from Gilburd that shows that in GBS, antibodies are made to phospholipids and the phospholipids also contain that phosphoglycerol," with "some of the strongest binding" being to phosphatidylserine, phosphatidylcholine, and phosphatidylethanolamine, which "are all major targets of the immune response" in GBS. (Tr. 308-09, 311.) Although Dr. Fujinami opined that it is impossible to know whether the suggested immune response is to the phosphoglycerol component in particular (*Id*. at 192, 195 (discussing Nakos et al., *supra*, at Ex. 55)), petitioner need not prove scientific certainty to establish entitlement to compensation in this Program. *Knudsen*, 35 F.3d at 548-49.

Accordingly, petitioner has preponderantly shown that the Prevnar 13 vaccine can cause the AIDP form of GBS. Because I have concluded that Dr. Steinman's theory of molecular mimicry based on phosphoglycerol is sound and reliable, I do not reach the additional question of whether Dr. Steinman's separate theory based on CRM197 is likewise sound and reliable.

b. *Althen* prong three

Under the third *Althen* prong, a petitioner must demonstrate a "proximate temporal relationship" between the subject vaccination and the alleged injury. *Althen*, 418 F.3d at 1278. To do this, petitioner must provide "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008) (citations omitted).

The parties do not dispute that onset of petitioner's GBS occurred within 9-12 days of her Prevnar 13 vaccination. (ECF No. 121, pp. 24-25; ECF No. 123, pp. 25 n.12, 56-57; ECF No. 132, pp. 5 n.7, 72-73; ECF No. 135, pp. 2, 31-32.) However, respondent disputes Dr. Steinman's reliance on a study by Schonberger et al., to establish petitioner's onset of her GBS as falling within a timeframe consistent with his theory of causation. (ECF No. 121, p. 25 (discussing Schonberger et al., *supra*, at Ex. 47).) Respondent contends that Dr. Steinman's reliance on the Schonberger et al. study "has two glaring defects." (*Id.*) First, the study looked at a completely different vaccine – the 1976 swine flu vaccine. (*Id.*) Second, and relatedly, the studied vaccine did not contain the alum adjuvant contained in the Prevnar 13 vaccine. (*Id.*)

Regarding the first point, respondent is correct in noting that the study involved a different vaccine than the one at issue in this case; however, the findings of this study have been utilized in assessing risk periods in subsequent studies that have looked at other potential causes of GBS. (Baxter et al., *supra*, at Ex. 46, pp. 6-7 (referencing the 1976 swine flu vaccine study as helpful for selecting an appropriate time interval); *see also* Haber et al., *supra*, at Ex. 58, p. 5 (noting that the study results "verified 11 GBS reports with symptoms onset within 42 days of [Prevnar 13] vaccination" and referencing the Schonberger study in the bibliography).) Other special masters have also referenced Schonberger et al. in evaluating whether petitioners, who have alleged that they suffered GBS following Prevnar 13 vaccinations, have met their burden of proof under the third *Althen* prong. *See, e.g.*, *Godfrey v. Sec'y of Health & Human Servs.*, No. 17-1419V, 2025 WL 896840, at *28 (Fed. Cl. Spec. Mstr. Feb. 26, 2025) (Dorsey); *Koller*, 2021 WL 5027947, at *22-23 (Gowen).

Regarding the second point, Dr. Steinman testified that his opinion regarding the role of the alum adjuvant contained in the Prevnar 13 vaccine is not an essential component of his causal theory. (Tr. 115-16, 118.) But in any event, respondent's argument is limited to merely noting the existence of this distinction. (ECF No. 135, p. 32.) He has not pointed to any medical evidence suggesting that the presence of an alum adjuvant is a meaningful distinction vis-à-vis the expected timing of onset. (*See id.*) On respondent's behalf, Dr. Fujinami explains that he cannot predict timing based on Dr. Steinman's proposed causal theories. (Tr. 248.)

However, Dr. Fujinami acknowledged on respondent's behalf that the presence of antiphospholipid antibodies within 3-4 days after induction of disease is consistent with the expected time frame for an adaptive immune response, though he does not agree they are causally relevant. (Tr. 200; *see also* Tr. 284-87 (discussing Kanter et al., *supra*, at Ex. 33).) The IOM report notes that, "[f]or may antigens the latency (lag phase) between primary exposure and development of the primary antibody response is 7 to 10 days," and while this time frame "is not specific to any particular antigen, it can be used as a reference point for the latency between antigen exposure and the initiation of some of the immune-mediated mechanisms described." (IOM Report, *supra*, at Ex. 71, p. 87.) Dr. Fujinami further acknowledged that the studies by Ho et al. and Kanter et al. support an experimental change in disease course 10 days following injection. (Tr. 207.) This understanding is consistent with Schonberger et al., in which the authors found increased risk of onset of GBS primarily within five weeks of vaccination with the 1976 swine flu vaccine. (Schonberger et al., *supra*, at Ex. 47.)

Accordingly, petitioner has preponderantly shown that onset of her GBS occurred within a medically acceptable time frame from which to infer vaccine causation under the third *Althen* prong.

c. *Althen* prong two

The second *Althen* prong requires preponderant proof of a logical sequence of cause and effect, which is usually supported by facts derived from petitioner's medical

records. *Althen*, 418 F.3d 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. Medical records are generally viewed as trustworthy evidence. *Cucuras*, 993 F.2d at 1528. These records are generally contemporaneous to the medical events and "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." *Id.* However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (reasoning that "nothing . . . mandates that the testimony of a treating physician is sacrosanct—that is must be accepted in its entirety and cannot be rebutted"). A petitioner may support a cause-in-fact claim through presentation of either medical records or an expert medical opinion. *See* § 300aa-13(a). The special master is required to consider all relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler*, 88 F.4th at 963 (citing *Hines*, 940 F.2d at 1528).

In his post-hearing brief, respondent stresses that petitioner must do more to meet her burden of proof under *Althen* prong two than simply rely on the other *Althen* elements, quoting the Federal Circuit as rejecting the notion that "proof that an injury could be caused by a vaccine and that the injury occurred within an appropriate period of time following the vaccination is sufficient to require an award of compensation." (ECF No. 135, pp. 28-29 (quoting *Hibbard v. Sec'y of Health & Human Servs*., 698 F.3d 1355, 1366 (Fed. Cir. 2012)).) Indeed, the *Althen* court held that "neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." 418 F.3d at 1278. However, the Federal Circuit has also explained that "a petitioner is certainly permitted to use evidence eliminating other potential causes to help carry the burden on causation and may find it necessary to do so when the other evidence on causation is insufficient to make out a *prima facie* case." *Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007). And, while respondent stresses that part of the Federal Circuit's *Capizzano* precedent that explains that the second *Althen* prong "is not without meaning," he omits the Circuit's further explanation that:

> [I]n our view, the chief special master erred in not considering the opinions of the treating physicians who concluded that the vaccine was the cause of Ms. Capizzano's injury. The fact that these physicians' diagnoses may have relied in part on the temporal proximity of Ms. Capizzano's injuries to the administration of the vaccine is not disqualifying. We see no reason why evidence used to satisfy one of the *Althen* [] prongs cannot overlap to satisfy another prong. In other words, if close temporal proximity, combined with the finding that hepatitis B vaccine can cause RA, demonstrates that it is logical to conclude that the vaccine was the cause of the RA (the effect), then medical opinions to this effect are quite probative.

440 F.3d at 1326 (internal citations omitted).

Here, there is no question that petitioner suffered from GBS following her January 15, 2015 Prevnar 13 vaccination. (Tr. 161; Ex. A, p. 5.) When she was first diagnosed by her treating neurologist as having GBS on January 27, 2015, the neurologist noted in the relevant history that "[s]he had recently had a pneumonia vaccine but otherwise has been in her usual state of health." (Ex. 10, pp. 79-80.) Thereafter, her primary care provider assessed "Guillain-Barre syndrome following vaccination" in the context of her post-inpatient follow up care. (*Id.* at 13-15.) "[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1280). And, while these notations are not detailed, "[o]rdinary medical care offers little beyond clinical history that would confirm the cause of GBS." *Bartoszek*, 2024 WL 4263604, at *23. The treater notations are also further buttressed by Dr. Steinman's opinion. *See* § 300aa-13(a)(1) (suggesting that petitioners may substantiate their vaccine claims with reference to medical records or the support of expert medical opinion). Based on a review of the medical records, Dr. Steinman likewise opined on petitioner's behalf that her Prevnar 13 vaccine caused her GBS. (Tr. 34, 36-37.) In addition to presenting a theory of causation, Dr. Steinman further explained that there is no evidence of any preceding infection or other potential alternative cause that could explain petitioner's condition. (*Id.* at 36-37, 155-56.) Dr. Chaudhry likewise opined on respondent's behalf that petitioner "belonged to one third of GBS cases where no antecedent infection is established." (Ex. A, p. 9.) And this, of course, is some evidence supporting petitioner's case. *Walther*, 485 F.3d at 1151.

Respondent maintains that the treater notations contained within petitioner's medical records are inadequate to support petitioner's burden of proof because they were not accompanied by a medical theory. (ECF No. 30, p. 7; ECF No. 135, p. 30.) However, "[a]ny expectation that treating physicians will record the precise biological theories behind their belief that a patient's condition was caused by a particular trigger is discordant with the reality of medical treatment." *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 667 (2011). Moreover, as indicated in *Capizzano*, it is not disqualifying if a treater's opinion is informed in part by temporality. 440 F.3d at 1326. Respondent's interpretation of the relevant notations is also clouded by his position as to *Althen* prong one. (ECF No. 30, p. 7 (arguing that, "[a]bsent a persuasive medical or scientific theory establishing that Prevnar vaccine can cause GBS as alleged by petitioner, any suggestion that the Prevnar vaccine did cause petitioner's GBS is mere speculation"); ECF No. 135, p. 30 (contending that "the Prevnar-13 vaccine does not have any known association with GBS").) However, for the reasons discussed above, there is a viable theory of causation. *See Capizzano*, 440 F.3d at 1326 (explaining evidence pertaining to the *Althen* prongs can overlap).

Respondent further stresses that, even if treating physician statements do constitute supportive medical opinions, they are not sacrosanct and can be rebutted.

(ECF No. 135, p. 30 (citing *Snyder*, 88 Fed. Cl. at 746 n.67).) Significantly, however, respondent does not raise any other treating physician opinion or any fact from petitioner's own clinical history that would rebut the assessment that petitioner suffered GBS related to her preceding vaccination. Instead, respondent raises only two broader arguments, neither of which is persuasive. First, citing Dr. Fujinami's report, respondent argues that "if Dr. Steinman's hypothesis were correct, many other tissues in the body would be targeted by the immune response to the Prevnar-13 vaccine, not just the myelin in the peripheral nervous system." (ECF No. 135, p. 30 (citing Ex. H, pp. 1-2).) Second, citing Dr. Chaudhry's report, respondent argues that only the AMAN variant of GBS has been shown to result from molecular mimicry, whereas petitioner suffered AIDP. (*Id*. at 30-31 (citing Ex. A, p. 5).) Respondent indicates that "Dr. Chaudhry observes that it is 'well established that ganglioside antibodies arising as a result of molecular mimicry' can cause the AMAN form of GBS." (*Id*. at 31 (quoting Ex. E, p. 2).)

Dr. Steinman explained, however, that gangliosides are present in both the central and peripheral nervous system. (Tr. 7, 76.) Yet, AMAN, which both Dr. Steinman and Dr. Chaudhry agree results from molecular mimicry-related ganglioside antibodies, is likewise a peripheral nerve disorder. (*Id.*; Ex. E, p. 2.) Thus, Dr. Chaudhry's emphasis on molecular mimicry in AMAN undercuts Dr. Fujinami's criticism. However, medical literature accompanying Dr. Chaudhry's report also explains that "a wider range of immune stimulants cause acute inflammatory demyelinating polyneuropathy compared with acute motor axonal neuropathy," and that "a wider range of anti-nerve autoantibodies directed at both proteins and glycolipids could be responsible for acute inflammatory demyelinating polyneuropathy immunopathology than is the case for acute motor axonal neuropathy." (Willison et al., *supra*, at Ex. A, Tab 1, p. 4.) Therefore, Dr. Chaudhry's suggestion that molecular mimicry is relevant only to the AMAN variant of GBS is also not persuasive.

The *Capizzano* court explained that a petitioner who satisfies *Althen* prongs one and three may nonetheless fail to satisfy *Althen* prong two where "medical records and medical opinions do not suggest that the vaccine caused the injury, or where the probability of coincidence or another cause prevents the claimant from proving that the vaccine caused the injury by preponderant evidence." 440 F.3d at 1327. These are not the circumstances of this case, even if the evidence specific to *Althen* prong two is relatively thin. Petitioner has preponderantly demonstrated a logical sequence of cause and effect establishing that the Prevnar 13 vaccine did cause GBS in this case.

d. Factor unrelated

Once petitioner has satisfied his own burden of proof, the burden shifts to respondent to demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to vaccination. § 300aa-13(a)(1)(B); *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367-69 (Fed. Cir. 2013). In this case, respondent has not offered any other factor as a potential cause of petitioner's GBS. (ECF No. 121, pp. 25-26; ECF No. 135, p. 32.)

VI. Conclusion

After weighing the evidence of record within the context of this Program, I find by preponderant evidence that petitioner suffered GBS caused-in-fact by the Prevnar 13 vaccination she received on January 15, 2015. A separate damages order will be issued.

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master